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OESOPHAGEAL HIATUS HERNIA OF THE CARDIAC END OF THE STOMACH

ASSOCIATED WITH A SECOND OESOPHAGEAL HIATUS HERNIA CONTAINING OMENTUM

REPORT OF AN UNUSUAL CASE

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The patient, a female aged 74 years, complained of intermittent attacks of upper abdominal pains of three years' duration.

History. These attacks started three years before. The subsequent attacks were similar in nature but increased in severity as time went on. The pain came on only when she was recumbent, usually between 11 p.m. and 4 a.m. commenced in the right hypochondrium and radiated over the epigastrium to the left side. It did not radiate to the shoulders or to the back. The pain was of a constrictive nature and it felt as though a band was drawn tightly across the body. At the same time there was severe discomfort in the chest as though the heart were pushed aside. Such an attack would last for anything from half an hour up to four hours. Deep breathing aggravated the pain.

During an attack she would vomit quantities of mucus but never food. At no time was there any haematemesis. At first the periods of remission from pain would last from four days to three weeks. Latterly, however, the periods of remission had become shorter and the pain had become progressively worse and lasted longer, with the result that she had little sleep at night. The pain had not been relieved by dietetic measures or the taking of alkalis.

X-ray. A diaphragmatic hernia was noted ('A' in Fig. 1) and the oesophagus appeared rather short. The duodenum was normal.

Operation. After her physical state had been assessed and her fluid and electrolyte balance attended to, she was operated upon on 3 August 1949.

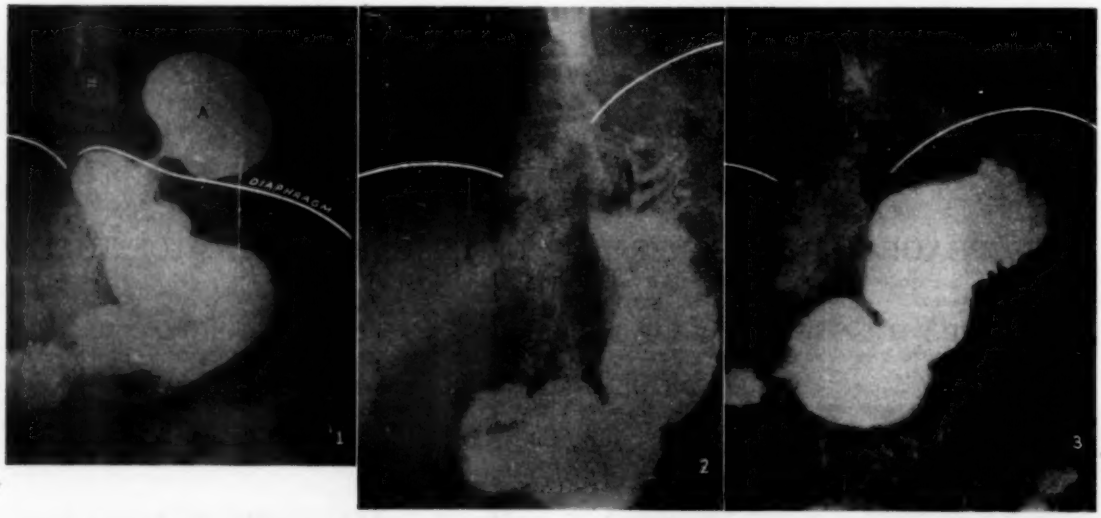
After the method described by Richard Sweet of Boston[1], with the patient lying on her right side, the left eighth rib was resected, the pleura incised and the rib spreader inserted. The inferior pulmonary ligament was freed and the lower lobe of the left lung retracted. The mediastinum, with its contained hernia, was now clearly visible. The left phrenic nerve was crushed at the point where it left the pericardium on its way to the diaphragm.

Next the mediastinal pleura was incised longitudinally, the edges retracted and the margins of the oesophageal hiatus bared of the attached diaphragmatic pleura. With blunt scissors dissection the sac (of what was thought to be the offending hernia of the cardiac end of the stomach) was exposed. It was soon realized, however, that this sac was, in fact, that of a second oesophageal hiatus hernia, because the oesophagus was seen not to enter this sac but to be lying on the posterior aspect of this sac. In the X-ray the oesophagus was seen to enter the offending hernia. The sac in question was freed completely from the surrounding structures and opened. It was a large sac, measuring about three inches in length and an inch and a half across, and it contained a large mass of omentum. There was a fairly tight constriction at the neck of the sac. With some diffieulty the contents were reduced into the peritoneal cavity and the excess of sac excised. The remainder was closed by means of interrupted sutures of fine black silk.

It was now apparent that the hernia actually causing the symptoms, viz. the oesophageal hiatus hernia of the

cardiac end of the stomach, was situated behind and to the left of the hernia already dealt with. The lower end of the oesophagus and the related right and left vagus nerves were freed by dissection. A Penrose drain was then placed around the lower end of the oesophagus and, whilst gentle traction was applied to it, the sac of the offending hernia with its contained portion of the stomach, was freed from the surrounding

During the operation the collapsed left lung was expanded by the anaesthetist (Dr. Brian Cockcroft) at intervals of about 20 minutes. At the completion of the operation, and just before final closure of the skin, the air in the left half of the thoracic cavity was aspirated by means of a catheter, introduced before, and attached to an Asepto syringe. At the same time the lung was fully expanded.



structures (Fig. 1). As is usual with this type of hernia, the wall of the stomach formed part of the sac, i.e. it was a sliding type of hernia. Without opening it, this sac was dealt with by inserting interrupted plicating sutures in the free peritoneal portion of the sac, using No. 3 zero black silk. These sutures proved adequate to reduce the hernia well below the level of the margins of the oesophageal hiatus. It was now evident that the shortness of the oesophagus, shown on the X-ray, was apparent rather than real.

Next, the margins of the oesophageal hiatus, which was greatly stretched as a result of its two contained herniae, were approximated about the oesophagus by means of interrupted sutures of No. 3 black silk. No difficulty was experienced in approximating the margins on the right side of the oesophagus. With the margins on the left side of the oesophagus, however, some difficulty was encountered because of the greatly attenuated margins and the intimate relationship of the aorta to them. The difficulty was overcome, however, and the margins were approximated. At the conclusion of this step in the operation the index finger could comfortably be introduced between the oesophagus and the newly constructed hiatus' margins.

Next the detached diaphragmatic pleura was re-attached to the margins of the hiatus, using interrupted sutures of No. 3 zero black silk.

The mediastinal pleura was re-sutured, using interrupted sutures of fine black silk, and the wound in the thorax closed in the usual way.

During the operation the patient received 1,500 c.c. of blood.

She was ambulant on the fourth post-operative day.

Since the operation the patient has been entirely free from pain.

An X-ray taken some weeks after the operation (Figs. 2 and 3) revealed that the cardiac end of the stomach had been reduced well below the level of the diaphragm, the latter being elevated.

The advantage of the transthoracic approach in this case was abundantly evident at the operation, in the comparative ease with which each of the two sacs was freed from the surrounding structures in the mediastinum, and in the way approximation of the margins of the oesophageal hiatus was possible under direct vision.

SUMMARY

A case of oesophageal hiatus hernia of the cardiac end of the stomach, associated with a second oesophageal hiatus hernia containing omentum, has been described.

The technique employed at operation has been fully described.

The advantage of the transthoracic approach is stressed.

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South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

THE SOUTH AFRICAN JOURNAL OF CLINICAL SCIENCE: A NEW ASSOCIATION JOURNAL

Clinical Proceedings, the Journal of the Cape Town Post-Graduate Medical Association, was established towards the end of 1941, since when it has built up a considerable reputation, indeed almost a tradition in the way of clinical journalism in South Africa.

With the advent of the weekly *South African Medical Journal*, however, the Cape Town Post-Graduate Medical Association recognized that a new situation had arisen requiring a change in policy as far as their own publication was concerned. It was felt that *Clinical Proceedings* should henceforth devote itself more exclusively to the advanced type of clinical research publication and, in order not to come into competition with the weekly *Journal*, the Post-Graduate Medical Association converted its own publication into a quarterly one.

The Medical Association of South Africa had itself for long recognized the need to cater for the more advanced type of medical writing and this was reflected in the policy of previous editors who produced a quarterly scientific number when the *Journal* was still a fortnightly publication.

As a result of amiable negotiations between the Cape Town Post-Graduate Medical Association, the South African Medical Association and other interested organizations, the position has now been reached whereby the task of publishing *Clinical Proceedings* in a new form will be undertaken by the South African Medical Association. This has been made possible by the generous action of the publishers of *Clinical Proceedings*, who have agreed that *Clinical Proceedings* together with all its advertising revenue should be incorporated as the nucleus of a new journal under the title of the *South African Journal of Clinical Science*. The South African Institute for Medical Research has joined as a third sponsor of its publication. The new journal will make its first appearance in March 1950.

We feel sure that the widely-based national support which the *South African Journal of Clinical Science* will now enjoy, is bound to ensure a substantial subscription membership for the new publication and we feel that all members of the Association will not hesitate to support this new undertaking.

VAN DIE REDAKSIE

DIE SUID-AFRIKAANSE TYDSKRIF VIR KLINIESE WETENSKAP: 'N NUWE TYDSKRIF VAN DIE VERENIGING

Clinical Proceedings, die Tydskrif van die Kaapstadse Na-graadse Mediese Vereniging, is teen die end van 1941 gestig vanaf watter tydstip dit 'n aansienlike naam vir homself verwerf het, inderdaad byna 'n tradisie in die kliniese joernalistiek in Suid-Afrika geskep het.

Met die koms van die weeklikse *Suid-Afrikaanse Tydskrif vir Geneeskunde* het die Kaapstadse Na-graadse Mediese Vereniging egter ingesien dat 'n nuwe toestand ontwikkel het, wat so ver dit hulle eie blad betref, 'n beleidsverandering genoodsaak het. Daar is gevoel dat *Clinical Proceedings* hom voortaan meer bepaald aan die gevorderde tipe kliniese navorsingspublikasie moet wy en, ten einde nie met die weeklikse *Tydskrif* mee te ding nie, het die Na-graadse Mediese Vereniging sy eie tydskrif in 'n kwartaalblad omgeskep.

Die Mediese Vereniging van Suid-Afrika het self al lank besef dat daar behoefte is aan voorsiening vir die gevorderde soort mediese artikel en dié besef is weerspieël in die beleid van vorige redakteurs wat 'n wetenskaplike kwartaalnommer van die *Tydskrif* uitgegee het toe dit nog elke veertien dae verskyn het.

As gevolg van vriendskaplike onderhandelings tussen die Kaapstadse Na-graadse Mediese Vereniging, die Suid-Afrikaanse Mediese Vereniging en ander belanghebbende organisasies, is 'n punt nou bereik waar die taak om *Clinical Proceedings* in 'n nuwe vorm uit te gee deur die Suid-Afrikaanse Mediese Vereniging onderneem sal word. Dit is moontlik gemaak deur die grootmoedige optrede van die uitgewers van *Clinical Proceedings* wat ingestem het dat *Clinical Proceedings* met al sy inkomste uit advertensies ingelyf word as die kern van 'n nuwe tydskrif met die naam *Suid-Afrikaanse Tydskrif vir Kliniese Wetenskap*. Die Suid-Afrikaanse Instituut vir Mediese Navorsing het as 'n derde borg vir die publikasie daarvan aangesluit. Die nuwe tydskrif sal sy eerste verskyning in Maart 1950 maak.

Ons voel oortuig dat die wydvertakte nasionale ondersteuning wat die *Suid-Afrikaanse Tydskrif vir Kliniese Wetenskap* nou sal geniet, ongetwyfeld 'n aansienlike aantal intekenaars vir die nuwe blad sal verseker en ons voel ootruig dat al die lede van die Vereniging sonder aarsel hierdie nuwe onderneming sal ondersteun.

For the time being this new journal will be published quarterly; but it is hoped that, as the necessary material submitted for consideration increases in amount, the new journal may revert to a monthly publication.

The subscription rate is the modest one of 25s. per annum and we hope that the members of the Association will respond readily to this invitation to subscribe. Cheques may be forwarded to: *The South African Journal of Clinical Science*, P.O. Box 643, Cape Town.

Vireers sal hierdie nuwe tydskrif elke kwartaal verskyn; daar word egter gehoop dat wanneer die nodige materiaal wat vir oorweging ingestuur word in hoeveelheid toeneem, die nuwe tydskrif in 'n maandblad omgeskep sal kan word.

Die intekengeld is op die oomblik slegs 25s. per jaar en ons hoop dat die lede van die Vereniging geredelik op hierdie uitnodiging om in te teken sal reageer. Tjeks kan gestuur word aan: *Die Suid-Afrikaanse Tydskrif vir Kliniese Wetenskap*, Posbus 643, Kaapstad.

HAEMOPTYSIS IN MITRAL STENOSIS

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(*Continued from p. 97*)

5. THE PATHOGENESIS OF HAEMOPTYSIS IN MITRAL STENOSIS

The pathogenesis of the haemoptysis is intimately bound up with the pathological changes found. While as a rule mitral stenosis is the significant valvular lesion, other valvular lesions may be associated. In Wolff's and Levine's series of 50 cases,[4] 22 had multiple valve involvement. In the Johannesburg cases, two non-European and three European haemoptysis cases had multiple valvular lesions. Nevertheless, it may be assumed that the mitral stenosis is the significant lesion in the production of haemoptysis.

The probable course of events leading to pulmonary congestion in mitral stenosis is as follows: The narrowed mitral orifice limits the amount of blood which can reach the left ventricle. The latter can probably expel all the blood presented to it. Thus the pulmonary hypertension is not caused by back pressure due to actual failure of the left ventricle, but by an overstrong right ventricle which tends to flood or overcrowd the pulmonary circulation with blood. Emotion, exertion or tachycardia cause acceleration of the circulation, often aided by the milking of venous blood back to the heart by contraction of the skeletal muscles during exercise, and present the hypertrophied right heart with an increased amount of blood, which it in turn forces into the pulmonary circulation. The increased amount cannot in the same time pass the narrow mitral valve, and the pressure in the pulmonary vessels becomes greater than the vessels can stand and haemoptysis occurs. This same intense engorgement may lead to pulmonary oedema or cardiac asthma and the same patient may exhibit all these manifestations of pulmonary hypertension at the same or different times.[4, 14, 15, 20, 22] The controlling factors giving rise under one set of conditions to haemoptysis, and in another to pulmonary oedema characterized by extravasation from the blood stream of the fluid portion of the blood with a few laked blood cells, is not known.[14] The absence of very marked liver enlargement, ascites, oedema and undue venous engorgement shows that the syndrome is not caused by right heart failure. Furthermore, if failure of the right ventricle occurs subsequently, producing the above-mentioned signs, there is relief from the intense pulmonary congestion and dyspnoea. Fishberg[10] states that he has twice seen haemoptysis first appear in cases of mitral stenosis with auricular fibrillation, in whom digitalization had cleared up systemic venous stasis. A further proof of pulmonary congestion and hypertension in this condition is that patients often say that they feel better after an haemoptysis and that the dyspnoea is less.

Another less plausible explanation of the pulmonary congestion and hypertension is that the left ventricle is small or of normal size, and being unaccustomed to expel much blood because of underfilling, fails abruptly when suddenly supplied with an excess of blood, leaving the surplus behind in the lungs. This is unlikely to be the correct explanation.[20]

It is probable that the occurrence of haemorrhage depends not only on the degree of congestion and hypertension, but also on the presence of sclerosed vessels. which rupture under the strain placed on them.

Parker and Weiss[21] described certain changes in the lung vessels in cases of mitral stenosis, changes which may be of great significance in some instances of haemoptysis. They found that long-continued hypertension, stagnation and oedema, caused wide dilatation and apparent elongation of capillaries with bulging into the alveolar spaces. In some cases there was actually aneurysmal dilatation. The larger arteries showed proliferative changes in the intima, with the occurrence of lipoid-filled macrophages and increased connective tissue. The changes were analogous to the kidney lesions of systemic hypertension. In some cases, the arterioles showed hyperplastic arteriosclerosis or productive endarteritis similar to the changes in the renal arterioles in malignant hypertension. This similarity was further enhanced in rare cases by the occurrence

of necrotizing arteriolitis. Other authors have also described vascular sclerosis in the lungs in pulmonary hypertension.[23, 24] The first change to occur in progressive pulmonary engorgement is an increase in the number of the visible capillaries; later on dilatation occurs. The advanced vascular lesions can develop within about two months.[21] Parker and Weiss[21] consider profuse pulmonary haemorrhage to be due to rupture of the larger, bulging capillaries and express uncertainty whether the diseased arterioles can rupture. Other authors consider that haemorrhage can occur by diapedesis from the dilated capillaries.[13, 14]

Other writers have speculated on the possibility that active rheumatic infection of the pulmonary arteries may be responsible for haemorrhage.[4]

The sclerotic changes described above are by no means always present. Wolff and Levine[4] only found sclerosis of the pulmonary vessels in six (36%) of the autopsied cases. Ferguson *et al.*[9] after careful study found that two of their four haemoptysis cases with mitral stenosis showed no sclerosis. These authors came to the conclusion that in cases of mitral stenosis in which infarction and acute pulmonary oedema are not present, haemoptysis is probably due to bleeding from varices of the bronchial veins. They do not believe that pulmonary hypertension and sclerosis can lead to rupture of vessels or that sudden dilatation of lung capillaries with diapedesis into the alveoli, or similar dilatation with rupture of the capillaries lining the small bronchioles, is adequate to explain the haemoptysis. They rightly assert that it does not seem likely that diapedesis into the alveoli can account for sudden profuse haemorrhage. Normally, the blood flow is from the bronchial to the pulmonary circulation through communications between the pulmonary and bronchial veins. These authors believe that, in mitral stenosis, the flow to the left auricle through the pulmonary veins is hindered and the pressure in these veins rises above that in the right auricle. This causes a reversal of the blood flow through the anastomosis and the blood passes from the pulmonary into the bronchial veins and back to the right auricle via the azygos, hemiazygos and intercostal veins. The submucosal bronchial veins of the smaller bronchi leading towards the main bronchi lilate greatly to handle the increased flow, as shown by *post mortem* angiography, and even become grossly visible. Haemorrhage is probably due to rupture or ulceration of these engorged bronchial veins, resembling massive bleeding from oesophageal varices. A severe coughing spell, mild ulcerative bronchitis or a rise in left auricular pressure could easily initiate haemorrhage.

In some cases, haemoptysis may be due to pulmonary infarction, which is not uncommon in cases of mitral stenosis.[2, 4, 9] While the embolus may arise as usual from the systemic veins, in this condition it may also come from the thrombi in the chambers of the right side of the heart, especially in the presence of auricular fibrillation.[25] Levine and White[25] found no evidence of intracardiac thrombi in their mitral stenosis cases at autopsy, but Wolff and Levine[4] found right auricular thrombi in three of their cases. A further source of emboli in mitral stenosis is an acute or subacute infection on an old rheumatic valve. Haemoptysis due to infarcts in subacute *Streptococcus viridans* endocarditis has often been described.[25] Finally, primary thrombosis *in situ* in the smaller pulmonary arteries may be the source of infarction. This is probably favoured by pulmonary arteriosclerosis, and the sluggish pulmonary circulation due to pulmonary engorgement.[25, 26] It is a well-recognized fact that infarction usually occurs in the presence of congestive cardiac failure, unlike the previously described forms of 'pulmonary apoplexy.[4, 8, 25, 26].

6. PROGNOSTIC SIGNIFICANCE OF HAEMOPTYSIS

The immediate prognosis of haemoptysis in mitral stenosis is usually good. Fatal cases are, however, not unknown.[3, 12] Far more important than the immediate prognosis, however, is the prognostic significance of the haemoptysis. While some individuals never expectorate blood despite intense pulmonary engorgement, others have repeated attacks. A patient who has once had an attack tends to have it repeatedly.[10] Haemoptysis has long been recognized as a poor prognostic sign in mitral stenosis. Sir James Mackenzie, in the last edition of his book, states that 'haemoptysis is a grave sign, the patient dying sometimes shortly after an attack'; although in auricular fibrillation the patient may make a good recovery, especially after the use of digitalis.[16] Haemoptysis is the result of long-standing and marked mitral stenosis and thus indicates a very poor cardiac status. It is for this reason correlated with a poor prognosis. The present writer, by showing that the age incidence of the haemoptysis cases is lower than that of the cases without haemoptysis in the Johannesburg group, has contended that the haemoptysis cases have a shorter expectation of life than those without haemoptysis. This is borne out by other writers.[4, 13] Wolff and Levine[4] found the average duration of life following the initial haemoptysis to be 32.5 months in their cases, while the average length of life of cases without haemoptysis was 50 months from the time of admission to hospital. The average duration of life was two years or less from the initial haemoptysis in 75% of their cases and only five cases survived five years or longer. They conclude that none of the common complications of rheumatic heart disease, with the exception of subacute bacterial endocarditis, has such a serious prognostic implication. Three of their patients died within one or two days after the onset of haemorrhage. Such sudden death in young people is dramatic and unexpected. These patients may only have slight cardiac enlargement and no signs of failure or irregular rhythm, and should normally have an excellent prognosis. Patients with no cardiac enlargement at all do not appear to have such a poor prognosis. On the whole, these authors[4] found that the mortality was higher and the length of survival shorter than would have been predicted on the basis of degree of cardiac enlargement and congestive failure alone. They also found the mortality was higher among the patients with pulmonary infarction than in the series as a whole. This latter fact is supported by the work of Levine and White,[25] who mention that infarction in the presence

of congestive failure retards all forms of treatment for the failure.

In Oppenheimer and Schwartz's cases of paroxysmal pulmonary haemorrhage,[13] most of the patients died within two to three years after onset of the episodes. Ferguson's four cases[9] lived one day, four weeks, three months and nine years respectively after their initial haemoptysis, while Stewart's three cases[14] lived six months, four years and three months respectively.

Thus, while a true statistical analysis of a large series of such cases yet requires to be done, it appears from the evidence at present available that the occurrence of moderate to large haemoptysis in mitral stenosis is a grave prognostic sign.

7. TREATMENT

The treatment of pulmonary haemorrhage (apart from pulmonary infarction) in mitral stenosis has hitherto been entirely neglected by many authors, or glossed over as being unnecessary. This appears to be a serious omission, since haemoptysis is a manifestation of pulmonary hypertension, which in itself requires treatment. While the treatment in the presence of congestive failure is obviously the classical treatment of this latter condition, by means of digitalis, diuretics, salt-free diets and the like, the treatment of massive haemoptysis in the absence of congestive failure suggests a different problem.

Whether haemoptysis be due to an overstrong right ventricle causing pulmonary flooding or whether it be due to rupture of bronchial veins, the essential lesion is pulmonary congestion. An increase in cardiac action cannot force more blood through the narrow mitral valve, and must thus cause further pulmonary congestion and possibly intensify the haemorrhage. The use of digitalis with its effects on venous pressure and cardiac action is therefore debatable.[27] The cardiac output in these cases of mitral stenosis is variable[22] and any discussion of the effect of digitalis must necessarily be highly theoretical. McGinn and White[20] find digitalis and its products of less value in the treatment of cardiac asthma in mitral stenosis than in that form due to left ventricular failure and it is probable that the pathogenesis of haemoptysis and cardiac asthma in mitral stenosis is similar. Weiss[8] stresses that the use of digitalis and strophanthin are contra-indicated. The present writer has seen no benefit from the use of digitalis in a few cases and believes that intravenous mercurial diuretics, venesection of up to 1,000 c.c. of blood, or 'bloodless venesection' by means of tourniquets on the limbs, and the administration of oxygen should form the basis of treatment, as for cases of pulmonary oedema or paroxysmal dyspnoea. The value of morphine in moderate doses cannot be doubted.

In the past it has been recognized that cases of Lutembacher's syndrome of mitral stenosis combined with atrial septal defect, do not get acute attacks of pulmonary oedema and haemoptysis, and this has led to the recent attempts to create similar shunts in patients with extreme mitral stenosis, but with good heart musculature, who succumb from high pulmonary pressure—a state termed by Bland[28] as 'lung failure' in contradistinction to heart failure. Sweet[28] anastomosed the azygos vein to the dorsal segment branch of the inferior pulmonary vein in five cases, with improvement in four cases, in order to achieve this object, while Harken, Ellis *et al.*[22] have produced artificial interatrial defects in two patients with survival in each case, and have performed mitral valvuloplasty in two other cases, with one survival, in further attempts to relieve the pulmonary pressure.

There is no doubt that further work of this type will lead to a better understanding of the principles involved in the pathogenesis and prevention of haemoptysis and other manifestations of pulmonary hypertension.

Finally, in the prevention of haemoptysis in patients with mitral stenosis, advice on how to avoid the precipitating factors such as emotion, exertion and tachycardia, must not be neglected.

8. ILLUSTRATIVE CASES

The following case histories illustrate some of the features described in this paper:—

Case 1. Mr. R. B., a 37-year old European attorney, was first seen on 9 December 1945, complaining of 'coughing up blood'. He had been well until 1941 when, while on army manoeuvres, he noticed breathlessness on exertion. His medical officer then regraded him on account of a 'leaking valve'. From December 1944 he became increasingly breathless on exertion and gave up golf for this reason.

For the past two weeks he had had a cold with cough and nasal discharge. The cough produced scanty whitish sputum. On December 9, while walking, he started coughing and suddenly brought up half a cupful of bright red blood. On examination, he had a typical presystolic apical murmur and thrill. There was no obvious enlargement of the heart, no oedema of the legs, no liver enlargement and no engorgement of the neck veins. No adventitious sounds were heard in the chest. On bed rest, the haemoptysis ceased in three days and the cough in one week.

Following this, he was well until June 1946, when he developed 'bronchitis', which lasted for three days. On 27 July, following a large dinner, he went for a walk and became extremely breathless. At 10 p.m. examination showed extreme dyspnoea and difficulty in talking. He could not lie down, and even lifting his legs on to the bed increased the dyspnoea The pulse rate was 110 per minute and the blood pressure was 120/80 mm. Hg. The breath sounds at both bases were diminished, but no adventitious sounds were heard. The neck veins were obviously distended and the liver was just palpable. Cyanosis of the lips was present. A subcutaneous injection of morphine gr. ¼ and atrophine gr.1/100 produced temporary relief, but after an hour he became disorientated and extremely cyanosed, with cold extremities, an imperceptible pulse and coarse bubbling râles at the bases. Oxygen was administered but during a venesection, respiration suddenly ceased, and some pink frothy mucus appeared at the mouth. All attempts at resuscitation failed.

This case depicts well two aspects of pulmonary hypertension—haemoptysis induced by a fit of coughing and pulmonary oedema induced by exertion. It is typical that there were no obvious signs of congestive failure until the terminal episode.

Case 2. B. W., a male Bantu woodcutter, aged 30 years, was admitted to Coronation Hospital on 14 June 1946, complaining of 'swelling of the body for two weeks, coughing blood and breathlessness for six months'.

Since 1939 he has had bouts of coughing and breathlessness on exertion, becoming progressively worse. For the last six months he has had spells of 'coughing up blood', lasting two to three days up to one month at a time. The blood was bright red and not mixed with sputum. He often brought up a cupful of blood at a time and during an attack of haemoptysis his symptoms were aggravated and he had a 'tight feeling' in his chest. His legs and scrotum had become progressively more swollen in the last two weeks.

At the age of 19 years he had joint pains flitting from joint to joint and lasting for three months.

On examination, the heart was found enlarged to the left and the right by percussion, and the apex beat was palpable 4 inches from the mid-line in the fifth space. Systotic and diastolic murmurs were heard at the apex, the diastolic being rumbling and crescendo in character. A soft, blowing systolic murmur was heard over the pulmonic area and the left side of the sterum. Breath sounds were diminished at the bases, and moist sounds were present. The liver was much enlarged and ascites, oedema of the scrotum, thighs and legs, and over the sacrum was present. Radiographs suggested general cardiac enlargement with some 'mitralization' and pulmonary congestion.

There was nothing significant found in the routine laboratory tests. The electrocardiogram showed broad and notched P. waves in leads 1 and 2 with a PR interval in lead 2 of 0.24 second. There was right axis deviation. The blood sedimentation rate performed subsequently was increased.

On a salt-free diet, digitalis and diuretics the patient improved but developed a Wenkebach phenomenon in the electrocardiogram, presumably due to digitalis intoxication. Twice, because of this intoxication, digitalis was stopped and congestion and oedema recurred. On each occasion, starting the digitalis again was followed by relief of the signs of venous congestion and, interestingly enough, by severe haemoptysis. Finally, he was discharged three months later on a maintenance dose of digitalis.

This case has several interesting features. The prolonged PR interval and high sedimentation rate probably indicate active rheumatic disease which may have been the cause of some of the episodes of haemoptysis. This case also shows that severe haemoptysis can occur with severe congestive failure, as well as without failure. It is interesting that on two occasions, relief from venous congestion was followed by haemoptysis and that haemoptysis was accompanied by a 'tight feeling' in the chest.

SUMMARY

1. A group of 150 cases of mitral stenosis has been analyzed in regard to the incidence of haemoptysis.
2. The grave prognostic significance of haemoptysis has been indicated.
3. The clinical features and pathogenesis have been reviewed.
4. Modern views on treatment have been outlined.

I wish to acknowledge the kind permission of Dr. K. Mills to make use of the records of the Johannesburg Hospital.

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LOUSE- AND FLEA-BORNE TYPHUS

CEREBROSPINAL FLUID FINDINGS AND THE RESPONSE TO AUREOMYCIN AND CHLOROMYCETIN.

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Gear and Harrington[1] recently described two cases of tick bite fever treated with aureomycin. The present paper records certain findings and the result of treatment with aureomycin and chloromycetin in six of the acute cases of typhus fever recently admitted to the Isolation Hospital, Bloemfontein.

During September and October 1949 there was a wave of epidemic typhus fever in the Orange Free State. Ten cases were admitted to the Isolation Hospital, Bloemfontein, nine were non-Europeans from the same magisterial area, and one was a European from a different district. Of the 10 cases two died. The two deaths occurred on the tenth day of the illness. One of the deceased died eight hours and the other 48 hours after admission. Full investigations were not possible in the first case, but the patient showed the usual signs and symptoms of typhus, including the petechial rash and bloodshot eyes, and was unconscious at the time of admission.

In three cases referred to as A, B and C, the acute phase had subsided and aureomycin was therefore not given.

Case A. L. S.—A Native female aged 34 years was admitted on 30 September 1949, ill for eight days with headache, bloodshot eyes and fever. There was no rash at the time of admission. On the tenth day of illness the temperature was subsiding. On this day her blood agglutination was as follows:

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	±	—
OX K	+	—	—

The red cell count was 4,505,000 per c.mm.; the white cells 13,600 per c.mm. The differential count was: polymorphs, 63%; lymphocytes, 34%; monocytes, 1%; eosinophils, 2%. The blood also gave a positive field test for typhus prior to admission, using the S.A.I.M.R. test outfit. Blood Kahn, negative.

The results of the complement fixation tests were received on 26 October in respect of specimens taken on 1 October 1949.

R. prowazeki (epidemic typhus),	1 : 200
R. mooseri (murine typhus),	1 : 800
R. rickettsi (tick bite fever),	negative.
R. burneti (Q fever).	negative.

The result of this test, which was repeated and confirmed, indicates that this patient has had murine flea-borne typhus fever.

Case B. W. M. A Native female aged 32 years was admitted on 29 September 1949. She had been ill for about 11 days. The symptoms were similar to those of Case A. There was no rash on admission. The temperature of 100° F. subsided after 48 hours on 1 October, i.e. the twelfth day of the disease, the agglutinations were as follows:

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	—	—
OX K	±	—	—

The red cell count was 4,080,000 per c.mm.; the white cells 12,200 per c.mm. The differential count was: polymorphs 60%, lymphocytes 33%, monocytes 4%, eosinophils 3%. The blood Kahn was negative.

It is interesting to note that the patient took ill on 19 September, and on the seventh day of illness the field test was positive, whereas the S.A.I.M.R. Weil-Felix test was negative on that day. On 29 September the field test was more strongly positive, and the Weil-Felix test, too, was positive. The results of the complement fixation tests received on 26 October 1949 in respect of blood taken on 1 October were:

R. prowazeki (epidemic typhus),	1 : 800
R. mooseri (murine typhus),	1 : 6,400
R. rickettsi (tick bite fever),	1 : 25.
R. burneti (Q fever),	negative.

The result of the test which was repeated and confirmed, indicates that this patient has had murine flea-borne typhus fever.

Case C. S. S. A Native female aged 40 years, had been ill about 21 days with headache, fever, pain in the back and legs. She was admitted on 10 September with a temperature of 99.4° F., which thereafter remained normal. The patient was dehydrated with a dry coated tongue. There was no rash.

The field test for typhus was positive before admission. On 11 September (on the twenty-second day of the illness) the agglutination test was as follows:

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	±	—
OX K	±	±	—

On the twenty-fifth day of the illness the complement fixation test was as follows:

R. prowazeki (epidemic typhus),	1 : 400
R. mooseri (murine typhus),	1 : 50.
R. rickettsi (tick bite fever),	1 : 12.5.

The result indicates that this patient had epidemic louse-borne typhus.

The complement fixation tests were undertaken early in the outbreak to confirm our findings, so that the proper steps could be taken to deal with the correct vectors, namely lice and fleas. Unfortunately there was a long delay before the final reports were received—21 days—reducing somewhat their value in this connection.

The following records are those of the remaining six cases. Case 1 was from a different district, but Cases 2-6 were from the same area as the aforementioned Cases A, B and C.

The case records show in some instances the cerebrospinal findings and temperature and symptomatic response to aureomycin and chloromycetin.

In view of the spinal fluid findings which, in our cases, showed a polymorphnuclear as well as lymphocytic increase, typhus fever must be considered in the differential diagnosis of polioencephalitis, especially in cases where no rash is visible. The cost of treatment is also mentioned.

Case 1. W. W. A European male aged 28 years, was admitted on 5 September 1949 complaining of vomiting, headache, fever and pain in the back and limbs, of eight days' duration. Temperature, 104° F.; conjunctivae injected, and a typical typhus rash was presented. On the eighth day of the disease, the agglutination tests were as follows:

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	+	+
OX K	±	—	—

The end titre was:—

Proteus	OX 19,	1 : 1660.
Proteus	OX 2,	1 : 200.
Proteus	OX K,	1 : 25.

The complement fixation tests were:—

R. prowazeki (epidemic typhus),	1 : 200
R. mooseri (murine typhus),	1 : 25
R. rikettsi (tick bite fever),	1 : 12.5

This indicated that this patient had epidemic louse-borne typhus.

The blood Kahn was negative; the red cell count, 4,784,000 per c.mm.; the white cells 15,600 per c.mm.; the differential count: polymorphs 66%, lymphocytes 28%, monocytes 4%, eosinophils 2%.

On 7 September (the ninth day of the disease) the cerebrospinal fluid findings were 3 polymorph cells per c.mm. Chlorides, 610 mg. per 100 c.c.; sugar, 90 mg. per 100 c.c.; organisms, nil. Spinal fluid Kahn, negative.

On the eighth day of the disease, the patient was given aureomycin, 250 mg. 4-hourly; this was reduced to 250 mg. t.i.d. on 9 September, when the temperature had reached normal after about 60 hours, all symptoms having rapidly subsided. Total aureomycin given, 21 capsules of 250 mg.

Case 2. *I. S.* A Native female, aged about 28 years, was admitted on 30 September, complaining of headache, a stiff neck and pain in the limbs, with rash and fever of eight days' duration. On 1 October (the ninth day of disease) the agglutination tests were as follows:—

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	+	—
OX K	+	—	—

The complement fixation tests were as follows:—

R. prowazeki (epidemic typhus),	1 : 200
R. mooseri (murine typhus),	1 : 800
R. rickettsi (tick bite fever),	negative.
R. burneti (Q fever),	negative.

The result was repeated and confirmed indicating that this patient has had murine flea-borne typhus fever.

The red cell count was 4,776,000 per c.mm.; the white cell count, 13,200 per c.mm. The differential count: polymorphs, 66%; lymphocytes, 30%; monocytes, 2%; eosinophils, 2%. The field test for typhus was also positive before admission, using the S.A.I.M.R. test outfit. On admission the patient was gravely ill; temperature, 102° F. She was given chloromycetin 500 mg. (2 capsules) 4-hourly for 44 hours, without any change in her condition, and she died on 2 October. Total chloromycetin given: 22 capsules of 250 mg.

Case 3. *M. B.* A Native male aged 13 years was admitted on 23 September 1949 with symptoms of headache, fever, pain in the back and legs and a rash; duration four days. The patient's brother and sister from the same hut were admitted on the same day, with the same symptoms.

On 25 September (the seventh day of the disease) the agglutination tests were as follows:—

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	+	+
OX K	+	—	—

The red cell count was 4,144,200 per c.mm. The white cell count was 14,200 per c.mm. The differential count was: polymorphs, 66%; lymphocytes, 27%; monocytes, 4%; eosinophils, 3%. The rickettsial agglutination tests were:—

R. prowazeki (epidemic typhus),	1 : 200
R. mooseri (murine typhus),	1 : 200

The complement fixation tests were negative at this stage, and could not be repeated as the patient had been discharged when the result was received. The blood Kahn was negative.

On 27 September (the ninth day of the disease) the spinal fluid findings were 8 polymorphs, 1 lymphocyte and 1 monocyte per c.mm. Chlorides, 600 mg. per 100 c.c. Sugar, 58 mg. per 100 c.c. Organisms, nil. Spinal fluid Kahn, negative. On 26 September the patient was given aureomycin, 250 mg. 4-hourly, and received a total of 17 capsules. His temperature became normal after 72 hours, with a rapid improvement of all symptoms.

Case 4. *A. B.* A Native male aged 14 years, the brother of cases 3 and 5, was admitted on 23 September with headache, fever, pain in the back and legs, and a rash. Duration, four days.

The agglutination tests were as follows:—

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	+	+
OX K	+	+	—

The rickettsial agglutination tests gave the following results:—

	1 : 25	1 : 50	1 : 100	1 : 200	1 : 400
R. prowazeki	+	+	±	±	±
R. mooseri	+	+	+	+	+
	1 : 800	1 : 1600		1 : 3200	1 : 6400
R. prowazeki	—	±		+	—
R. mooseri	±	—		—	—

These indicate that the patient has had epidemic typhus, but as the differentiation was not clearly defined, a repeat test at a later date would have been advisable, but the patient had been discharged when this report was received.

On 25 September (the seventh day of the disease) the red cell count was 4,016,000 per c.mm.; the white cell count, 10,400 per c.mm. The differential count was: polymorphs, 68%; lymphocytes, 25% monocytes, 5%; eosinophils, 2%. The blood Kahn was negative.

On 27 September (the ninth day of the disease) the spinal fluid findings were: 9 polymorphs, 3 lymphocytes per c.mm.; organisms, nil; chlorides, 610 mg. per 100 c.c.; sugar, 61 mg. per 100 c.c. Spinal fluid Kahn, negative. Protein, increased.

Like his brother, the patient received 1 capsule (250 mg.) of aureomycin 4-hourly from 25 September. After 48 hours the patient's temperature had returned to normal and all symptoms had disappeared. He received a total of 17 capsules of aureomycin.

Case 5. *E. B.* A Native female aged 9 years, the sister of Cases 3 and 4, was admitted on 23 September, complaining of headache, fever, pains in the legs, a cough and a rash. Duration, four days.

The agglutination tests were as follows:—

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	+	+
OX K	+	+	—

The rickettsial agglutination tests gave the following results:—

R. prowazeki	1 : 200
R. mooseri	1 : 200

The complement fixation test was negative at this time, but could not be repeated as the patient had been discharged when this report was received.

On 25 September the blood Kahn was negative, and the red cell count was 3,928,000 per c.mm. The white cell count was 9,800 per c.mm. The differential count was: polymorphs, 70%; lymphocytes, 26%; monocytes, 2%; eosinophils, 2%. This was the seventh day of the disease.

On 27 September (the ninth day of the disease) the spinal fluid findings were polymorphs 25, lymphocytes

UMI

20, monocytes 2, per c.mm. Organisms, nil. Chlorides, 630 mg. per 100 c.c. Sugar, 51 mg. per 100 c.c. Spinal fluid Kahn, negative. Protein, increased.

The patient received aureomycin capsules, 1 (250 mg.) 4-hourly from 25 September. After 48 hours the temperature was normal and all symptoms had subsided. Total aureomycin given, 17 capsules.

Case 6. *L. F.* A Native female aged 28 years, was admitted on 6 October complaining of headache, fever, a rash, pain in the back, legs and arms. Duration, five days.

The agglutination tests were as follows:—

	1 : 20	1 : 100	1 : 200
OX 19	+	+	+
OX 2	+	±	—
OX K	±	—	—

The result of the complement fixation tests on a specimen of blood collected on 6 October 1949, was only received on 26 October, and was as follows:—

R. prowazeki (epidemic typhus),	1 : 50
R. mooseri (murine typhus),	1 : 400
R. rickettsi (tick bite fever),	negative.
R. burneti (Q fever),	negative.

The result of this test, which was repeated and the result confirmed, indicates that this patient had murine flea-borne typhus fever.

On 6 October (the fifth day of the disease) the blood Kahn was negative, and the red cell count was 4,128,000 per c.mm. The white cell count was 13,200 per c.mm. The differential count was: polymorphs, 66%; lymphocytes, 31%; monocytes, 2%; eosinophils, 1%. A lumbar puncture was not carried out.

This patient received chloromycetin, 2 capsules (500 mg.) 4-hourly from the time of admission; 44 hours later her temperature was normal. There was a slight rise on the next day, but it fell to normal soon after. All the other symptoms had subsided by this time.

SUMMARY AND COMMENTS

1. Ten recent cases of typhus fever are recorded.

2. The complement fixation and rickettsial agglutination tests were carried out by the South African Institute for Medical Research to establish the causal vector, so as to enable proper public health measures to be instituted.

3. Four cases, according to these tests, were murine flea typhus, and three louse-borne typhus. In one the rickettsial agglutination gave the same titre for both *R. prowazeki* and *R. mooseri*.

4. According to these tests both flea-borne and louse-borne cases occurred in the same epidemic.

5. The long delay before receipt of complement fixation reports reduced their practical value.

6. The cerebrospinal fluid findings are recorded. Because of the polymorphnuclear and lymphocytic changes in the cerebrospinal fluid, typhus fever must be considered in the differential diagnoses of polioencephalomeningitis.

7. Blood and spinal fluid Kahn tests are recorded. It will be seen that the Kahn test does not become positive in typhus fever, whereas textbooks state that the Wassermann test becomes positive.

8. Aureomycin and chloromycetin are both effective in both louse and murine typhus.

9. Aureomycin at the time of writing (21 October 1949) was costing the hospital 4s. 1d. per capsule wholesale—the retail price being approximately 6s. 2d. The price of chloromycetin was 3s. 2d. per capsule wholesale, and approximately 5s. 5d. retail. The total cost of treatment is therefore still fairly high.

10. The temperature drop only occurred after about 48 hours. These preparations will be most useful during the first ten days of the illness.

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BORNHOLM DISEASE

REPORT OF A CASE

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and
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This clinical entity is frequently misdiagnosed and confused with pneumonia, influenza, infectious mononucleosis, lymphocytic meningitis and acute surgical conditions of the abdomen. The diversity of clinical features easily accounts for this confusion.

Bornholm disease or epidemic pleurodynia is an acute febrile illness characterized by pyrexia, headache and paroxysmal pain in the region of the diaphragm. It is in the majority of cases a benign, self-limited disease without sequelae. Epidemic in nature, there are, however, many sporadic cases. The causal organism has not yet been isolated and little is known of the pathology of the disease other than that it involves a disturbance of the diaphragm and its overlying pleura. Young adults are especially affected and case reports indicate a high incidence in children. Scadding mentions the following criteria necessary for diagnosis:

i. Occurrence in epidemic form.
ii. Sudden onset with severe pleural pain.

iii. A brief but sometimes recrudescent febrile course terminating in complete recovery.

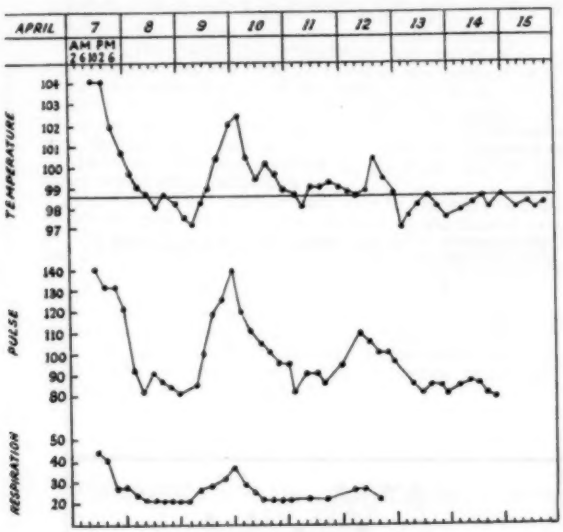
iv. Absence of evidence of an underlying pulmonary lesion.

The onset is, in the majority of cases, sudden; though prodromata such as malaise, sort throat and myalgic pains may rarely precede the characteristic symptomatology.

Pyrexia. Coincident with the onset of diaphragmatic pain the temperature rises to a peak of 102°-104° F. within the first 12 hours, thereafter gradually falling to normal in the next 12 hours.

Following a remission of 24-48 hours, there ensue one or more febrile recrudescences similar to the initial fever. The pulse rate is proportional to the temperature.

Respiratory System. Pain is the principal symptom, characteristically sudden in onset and pleuritic in nature, described as 'knife-like or stabbing'. It is usually felt over the lower ribs, located to the region of the diaphragmatic attachments and frequently referred also to the abdomen or shoulder. The duration of the pain is generally 1-2 days, occurring in paroxysms, then a remission of 2-4 days followed by a recurrence. Such symptomatic exacerbations may recur several times before recovery, the disease as a rule running a course of 5-10 days. A dry cough of brief duration is usually associated.



Diminished movement of the chest is evident during the paroxysms of pain. A pleural friction rub is present in 25% of cases, of fleeting duration and confined to the lower part of the chest on the lateral or posterior aspect. Cutaneous hyperaesthesia over segmental areas D9-11 may aid in dlfferentiation from an acute appendicitis. There is a striking absence of physical signs relative to the lungs.

Abdomen. Abdominal tenderness and rigidity may so predominate as to suggest the diagnosis of an acute abdomen. The site of tenderness is, in the majority of cases, the right hypochondrium though it may at times present in the right iliac fossa, the peri-umbilical region or the entire abdomen.

A transient sphenomegaly has been reported in a few cases.

Investigations: 1. *White Cells.* There is a progressive leucopenia most marked on the third and fourth day. Eosinophilia and as many as 11% atypical lymphocytes have been described.

2. *Red Cells.* There is no anaemia.

A slight rise in the *sedimentation rate* is observed in the early days of the disease.

Radiological examination of the chest reveals normal lung fields and normal diaphragmatic contours.

The *electrocardiogram* is normal in the majority of cases; a small percentage shows minor T-wave changes.

Complications: *Meningo-encephalitis.* Severe headache, drowsiness and slight neck rigidity may usher in this complication. The cerebrospinal fluid reveals a pleocytosis with 98% lymphocytes but normal protein and sugar. The culture is sterile. Pericarditis and orchitis are rare.

CASE REPORT

The patient was a 7-year-old girl who had been healthy and normal until 7 April 1949 when she suddenly complained of pain over the lower and lateral aspect of the right side of the chest, severe peri-umbilical pain and slight headache. The pain occurred in paroxysms and was unassociated with vomiting. A mild cough was present which aggravated the pain.

Physical Examination. The child appeared acutely ill. Temperature 104° F. Pulse 140 per minute; respirations, 40 per minute. Movement of the alae nasi was exaggerated and respiration obviously shallow on account of the pain. Hyperaesthesia was present over the lower and lateral aspect of the right side of the chest extending to the right hypochondrium. A coarse friction rub was heard over the right base; there were no other physical signs in the lungs. The heart was normal in size, with no murmurs.

Abdominal examination revealed generalized tenderness and voluntary guarding. However, as the paroxysm eased off there was elicited only questionable slight tenderness on deep palpation in the right upper quadrant. There were no palpable masses. Neurological examination was negative. The ears, nose and throat were unremarkable.

Course. The pyrexia subsided in 18 hours with marked clinical improvement, the child sitting up in bed and reading. Physical examination was completely negative.

A remission of 36 hours was followed by a repetition of the identical clinical picture, the temperature rising to 102° F. with rapid shallow respirations and the child again complaining of severe pleural and abdominal pain with voluntary guarding of the abdominal muscles. No physical signs were elicited in the chest or abdomen. This episode resolved in 18 hours.

The following investigations were performed:

Blood Count. (Third day of the illness at the height of pyrexia):— Red bloodcells, 4.5 million per c.mm.; white bloodcells, 6,900 per cmm. with a normal differential count.
Blood smear: Negative for malaria.
Urine: Nothing abnormal was found.
Chest X-ray: Clear lung fields with a normal contour of the diaphragm. Three days later another relapse ocurred, the temperature rising to 100.4° F. with only slight pain in the chest and abdomen.
Thereafter the child made an uneventful recovery.

During this same period one of us (M. H.) observed a strikingly similar case at the same school. This child was in the class of our patient and presented with only one febrile recrudescence after the initial pyrexia.

SUMMARY

A case of Bornholm disease in a 7-year-old female child is reported.

A brief review of the literature is presented with special reference to the clinical features.

We wish to express our gratitude to Prof. F. Forman for reviewing this article.

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ENTERIC FEVER

REPORT OF A CASE TREATED WITH CHLOROMYCETIN

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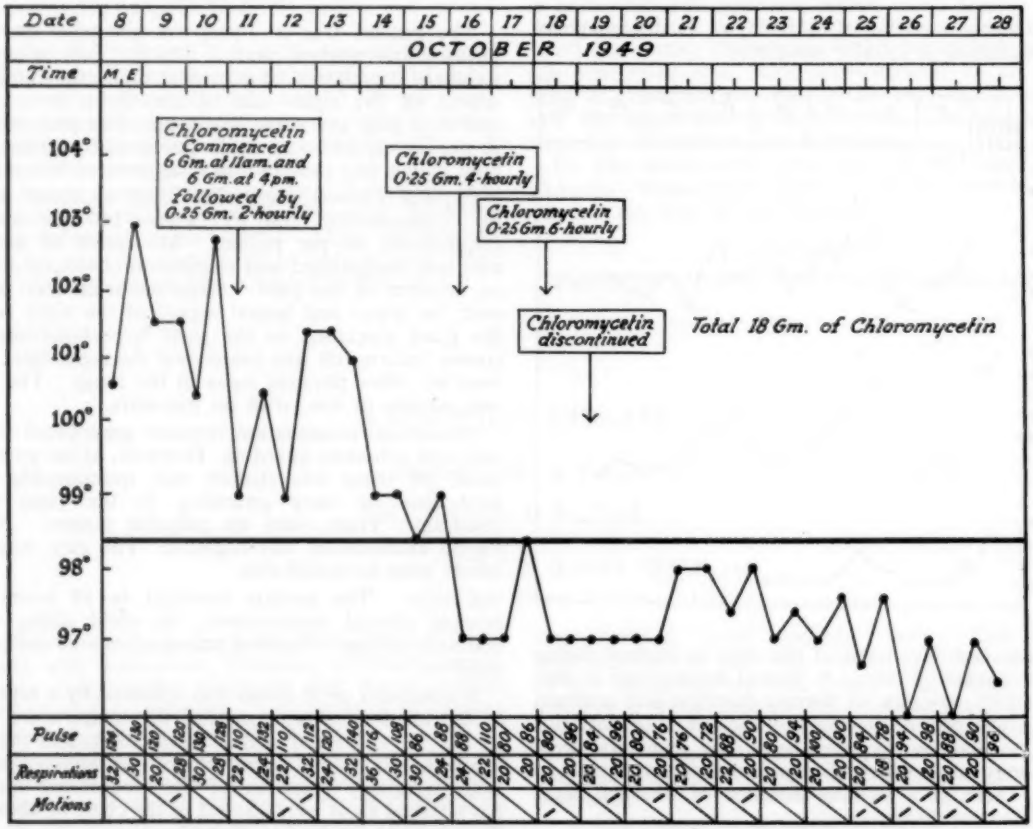
As there have been few published reports of cases of enteric fever treated with chloromycetin in South Africa, it was thought worth while recording this case.



Mrs. J. R., a 25-year-old European female, was admitted to the Ladysmith Provincial Hospital on 8 October 1949 with a history of having had a temperature for the past 13 days.

On 25 September she started having rigors with severe bouts of coughing followed by vomiting. Her doctor, who admitted her, reported that the vomiting was a prominent feature, but that there was little or no headache and no diarrhoea. She had been in bed at home before coming to hospital.

Physical Examination. Physically the patient was well

developed and appeared to be quite rational. She was seven months' pregnant. She was flushed and had a temperature of 101° F. Her mouth was dry and there were sordes on the lips. There was slight injection of the throat.

Lungs. There were ronchi over both lung fields but no other physical signs of note.

Heart. Blood pressure 130/80 mm. Hg. The heart was not enlarged. There was a systolic murmer over the apex.

Abdomen. The uterus was enlarged to the size of a seven months' pregnancy. The spleen was enlarged four fingers below the costal margin and the liver was palpable. There was no rash and no other physical signs. The foetal heart was heard in the right iliac region.

Past History. The patient stated that she had had enteric fever as a child. She has three children alive and has had one miscarriage. A tentative diagnosis of enteric fever was made and the following investigations were done.

10 October 1949. Blood Count: Hb, 62%; red cells, 3,350,000 per c.mm.; white count, 15,700 per c.mm.; band forms, 8%; polynuclears, 82%; lymphocytes, 9%; monocytes, 1%; eosinophils, 0%.

Urine: Straw coloured. S.G., 1010. No albumin and no sugar.

11 October. Stool: No pathogenic organisms were isolated on culture.

On the morning of 11 October the patient had become more drowsy and irrational, so chloromycetin was commenced. The initial dose was 6 gm. by mouth followed by 6 gm. at 4 p.m. and then 0.25 gm. two-hourly day and night.

The following day the blood culture report was positive for *B. typhosus.*

X-ray of the chest was normal and a Widal test done on 14 October read as follows: 'The Serum agglutinated the following organisms to the titre shown; *Salm. typhi*: 0, 1:200; H, 1:400.

The response to the chloromycetin was quite dramatic. Within 24 hours the temperature had dropped to 99° F. and clinically the patient appeared to be much better.

Within four days the temperature had returned to normal and remained normal. On 16 October the dose of chloromycetin was reduced to 0.25 gm. every four hours and on 18 October to 0.25 gm. every six hours. The patient received altogether 18 gm. of chloromycetin. On 19 October the patient was demanding to get up but she was only allowed up on 22 October. She was allowed to go home on 28 October, three weeks after admission.

Comment. The rapid return of this patient's temperature to normal plus the rapid improvement in the mental condition of the patient was quite dramatic. She reported that she felt foetal movements although at times the foetal heart could not be heard. When the patient was discharged the foetal heart could be heard easily.

Summary. A case of enteric fever in a seven months' pregnant woman is described. Treatment was started at least 16 days after the onset of the disease with a new antibiotic chloromycetin. The patient became afebrile within four days and symptomless within a week.

VERENIGINGSNUUS : ASSOCIATION NEWS

HOSPITAL ACCOMMODATION

MEMORANDUM FROM THE MEDICAL ASSOCIATION OF SOUTH AFRICA (CAPE WESTERN BRANCH) ISSUED 20 JANUARY 1950

For some considerable time the Medical Association of South Africa, Cape Western Branch, has been gravely perturbed about the inadequacy of hospital accommodation in the Cape Peninsula.

In February 1938 the then Governor-General stated, when opening the Groote Schuur Hospital, that even with the number of beds provided by that hospital we were still 10 years behind in the hospital accommodation required for the population. At that time the population of the Cape Peninsula (Divisional and Municipal areas) was approximately 374,520. To-day, 11 years later, it has increased by 157,480, and in the interval the number of additional hospital beds provided has been entirely insufficient to meet the needs of this increase. To-day it is often impossible to obtain the admission to hospital of patients who are acutely ill and for whom hospitalization offers the only chance of recovery. All medical practitioners, particularly those serving the poorer sections of the community, can tell of many instances where they have made unsuccessful attempts to obtain the admission to hospital of an acute surgical or medical case, and where failure to obtain a bed in some cases directly contributed to the death of the patient. The position to-day, therefore, has greatly deteriorated since the admittedly inadequate hospital accommodation of 1938.

From time to time representations in regard to the serious shortage of hospital beds have been made to the Provincial Administration by the Cape Hospital Board and the Medical Association. Although these have been courteously and sympathetically received, little or no improvement in the position has been achieved. Recently a deputation from the Medical Association was told by the Provincial Administration that the number of hospital beds available in the Cape Peninsula compared very favourably with that in any other part of the world.

The practice of modern medicine frequently involves intricate scientific investigation, facilities for which are available only at hospitals of the type of Groote Schuur. As a result, Groote Schuur Hospital is constantly being called upon to serve the needs of the sick from far outside the environs of the Cape Peninsula. This hospital should therefore be regarded as an institution which not only provides for the needs of the local patients but also of those living in other parts of the Cape Province. This fact should be taken into consideration when local requirements are being assessed. We consider that as medical men we are best qualified to express an opinion on the shortage of beds in the Cape Peninsula, and that it is our duty to focus attention on a situation which we regard as deplorable. All branches of medical practice in their efforts to relieve the urgent physical ills of the community are grossly hampered by the lack of adequate hospital accommodation. The orthopaedic and maternity services may be quoted as examples.

In 1948 orthopaedic surgeons in Cape Town stated that the position in respect of beds and out-patient facilities for their cases was desperate, and that with the existing inadequate accommodation it was impossible to carry out the continuous supervision and treatment of the young cripple to the age of adolescence necessary to prevent relapse or deformity. A partial solution of this problem was found by decentralizing the work and by using the Municipal and Cape Divisional Council Clinics. It is estimated that there are approximately 800 orthopaedic cases awaiting hospital treatment and that at the present rate of admission, viz. 60-70 cases per annum, it will take at least ten to twelve years to catch up with this backlog of patients. This does not take into account the number of new cases presenting for treatment during this period.

In regard to maternity cases there has been a small increase latterly in the number of beds, but the position is still far from satisfactory. As recently as 1947 attention was tragically focussed on the acute shortage of beds at the Peninsula Maternity Hospital, a teaching hospital, when an outbreak of virulent infantile enteritis occurred involving some 83 newly born babies and causing the death of 41—a case mortality of 50%. This hospital was designed for the accommodation of 40 patients, but owing to the lack of maternity beds it was frequently necessary to admit nearly double that number and also to accommodate two new-born babies in one cot. Undoubtedly this overcrowding was the main factor in precipitating and disseminating the disease.

The Medical Association realizes that the present financial stringency militates against the early provision of hospitals of a permanent type, and for this reason, and because of the urgency of the matter, the Association recently recommended to the Provincial Administration as an emergency measure, the erection of temporary hospital accommodation of the army type. Such accommodation proved very successful during World War II, and would effectively and expeditiously meet the immediate, pressing need for additional beds. Apparently,

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this expedient has not found favour with the Provincial Administration.

The Medical Association feels that the public should be made aware of the acute shortage of hospital beds in the Cape Peninsula and of the unnecessary suffering and despair which this is causing, and which could be relieved, for the time being, by the early erection of a temporary hospital of 500 beds.

PASSING EVENTS

Notification has just been received of the approval by the Secretary of State, of the promotion of Dr. M. L. Freedman to be Director of Medical Services of the Bechuanaland Protectorate with retrospective effect from 6 June 1949.

SIXTH INTERNATIONAL CONGRESS OF RADIOLOGY : LONDON, 1950

A copy of the preliminary programme, together with the necessary forms for completion, has been sent to all members of radiological societies throughout the world. Those planning to attend are urged to register as members of the Congress as soon as possible.

There are three classes of members: Full Members (including Junior, for those under 30); Associates accompanying Full Members, and Scientific and Technical Associates. All are reminded that a higher fee will be charged for registrations received after 1 April.

Members wishing to read papers must notify the Secretary-General, and an abstract should reach him by 1 April. Those who have a scientific exhibit to offer must also send details.

Application to join one of the tours of Great Britain and Ireland which are being organized for the week following the Congress must be received as soon as possible.

All communications should be sent to the Secretary-General, Sixth International Congress of Radiology, 45 Lincoln's Inn Fields, London, W.C.2.

Additional copies of the preliminary programme can be obtained from the same address on request, stating the language in which it is desired.

RHEUMATOLOGY PRIZE

INTERNATIONAL COMPETITION FOR AN UNPUBLISHED ARTICLE ON RHEUMATIC DISEASES

Under the Patronage of the 'Società Italiana di Reumatologia'

The Azienda Autonoma of the health resort of Acqui (natural mud baths) announces an international competition for an original and unpublished medical article dealing with the physiopathology, the clinical history and the therapy of rheumatic and arthritic diseases.

The competition is free and is open to all Italian and foreign doctors. The prize of 1,000,000 Italian liras will be awarded, by the international judging commission, for the best article, or may be divided between two articles.

Articles may be written in the Italian, French, English, Spanish and German languages.

The work or works awarded will be published under the care of the Azienda Autonoma di Cura of Acqui. The competition will close on 31 December 1950.

For detailed information and the rules of the competition, those interested should write to the Azienda Autonoma Di Cura Acqui, Piemonte, Italy.

SECOND EUROPEAN CONGRESS OF THE NATIONAL ASSOCIATION OF GASTROENTEROLOGY

This Congress will be held in Madrid from 4-7 May 1950. The general theme of the Congress is a discussion of *Diseases of the Biliary Tract, except Lithiasis and Cancer.* Representative contributions will be made by colleagues from America, the United Kingdom as well as Europe, including Scandinavia. Those who wish to attend this Congress are advised to send their applications to the General Secretary, Jorge Jua 41, Madrid, not later than 1 March 1950. The subscription rate for full membership and form of payment will be annouced in due course.

All members will have the right to take part in the discussions and present papers on the general theme. One session will be devoted to the reading of papers dealing with selected subjects. These must not exceed 600 words.

Members of the Congress, and their families enrolled as Associate members, will be invited to visit Toledo and El Escorial in an excursion arranged by the Organizing Committee. The ladies will be accompanied by the Ladies' Committee to visit museums and artistic monuments. A farewell banquet will be given to all members of Congress.

QUESTIONS ANSWERED

SIGNS OF DROWNING : DIFFERENTIAL DIAGNOSIS

Q. In what other post-mortem condition, besides death by drowning, is there present round the lips and nostrils fine white froth, resembling soap suds, which can also be made to exude from the mouth by pressure on the chest?

A. In cases of gross putrefaction the pulmonary decomposition is so severe as to make it possible for frothy fluid to appear at the nostrils and the mouth. This condition is, of course, easily recognizable by the markedly bloated and decomposed appearance of the body.

Any condition producing severe pulmonary oedema can also lead to appearances, indistinguishable from those seen in drowning, at the mouth and the nostrils. The writer has seen the classical, fine, frothy fluid presenting at the mouth and nostrils in cases of death from congestive cardiac failure. In these cases the patient literally drowns in his own secretions, so the autopsy findings are quite understandable.

In the case of congestive cardiac failure referred to, pressure on the chest increased the amount of froth appearing at the mouth and nostrils.

It should also be borne in mind that death accompanied by massive pulmonary oedema, from whatever cause, may result in the appearance of a fine froth at the mouth and the nostrils particularly if, after death, the head and the chest remain in a dependent position relative to the rest of the body.

REVIEWS OF BOOKS

PHILATELIC ENCYCLOPAEDIA

The Encyclopaedia of British Empire Postage Stamps (1806-1948). *Vol. II. The Empire in Africa.* (Pp. 448 + xvi. With illustrations and diagrams. 21s.) London: Robson Lowe Ltd. 1948.

Contents: Part I. South. Part II. Central. Part III. East. Part IV. North. Part V. West. Part VI. The African Islands. Appendix.

This volume will be of great interest to the large numbers of medical practitioners who are enthusiastic philatelists. The title of the volume is not misleading, as the scope of the information is genuinely encyclopaedic.

Of particular interest to philatelists in South Africa is the comprehensively detailed account given of the stamps which have come from what is now the Union of South Africa.

Profusely illustrated and thoroughly indexed, the volume is a memorable philatelic history of the provinces before Union as well as of South Africa since 1910.

The thoroughness with which the South African data have been documented is typical also of the other regions dealt with.

MEDICAL RECORDS

Manual for Medical Records Librarians. By Edna K. Huffman, R.R.L. (Pp. 371 + xxii, with illustrations. $4.50.) Chicago: Physicians' Record Co. 2nd ed. 1948.

Contents: Section I. The Functions of the Medical Records Department. 1. History of Medical Records. 2. The Medical Record—Its Analysis, Use and Value. 3. The Medical Records Librarian. 4. Responsibility for Securing the Medical Record. 5. Assisting the Physician in Securing Data. 6. Nurses' Notes. 7. Numbering and Filing Medical Records. 8. Storage of Medical Records. 9. Nomenclatures. 10. Cross-Indexing Procedures. 11. Statistics and Reports. 12. Group Studies. 13. Jurisprudence for the Medical Records Librarian. 14. Organization and Management. 15. Interdepartmental Relationship.
Section II. Medical Terminology. 16. Medical Terminology.
Section III. Secondary Duties of the Medical Records Librarian. 17. Correlation of Medical Reference and Medical Records Libraries. 18. Secretarial Duties.

The first edition of this manual was published in 1941, and in this, the second (1948) edition, innumerable alterations, additions and deletions have been made to make it comprehensive and modern.

The history of medical records is briefly traced from the Egyptian period to the twentieth century.

The chapters on Organisation and Management, Nomenclatures and Cross-Indexings and the sections on Microfilming and Punch Card Methods of Indexing and Cross-Indexing have been re-written to conform with recent advances and present-day requirements in the keeping of medical records. The chapter on Medical Terminology has been brought fully up to date. A new procedure in the record departments of some hospitals is discussed—the compilation of a Medical Illustrative Index.

The illustrations, charts and forms are well reproduced. Several years of experience as an instructor, the background of authority as past president and her membership on the Educational Board of the American Association of Medical Record Librarians give the author ample qualifications to have compiled this manual.

This book will be valuable for reference in hospitals and clinics, for use by medical records librarians and their assistants whose training has been through experience alone. It should be a potent influence in raising the standard of records to a high level.

BRAIN TUMOURS : DIAGNOSIS

Diagnosis and Treatment of Brain Tumors and Care of the Neurosurgical Patient. By Ernest Sachs, A.B., M.D. (Pp. 552. With 348 illustrations and 10 colour plates. $15.) St. Louis: The C. V. Mosby Company. 2nd ed. 1949.

Contents: 1. Surgical Anatomy and Physiology. 2. Methods of Examination. 3. The Surgical Pathology of Brain Tumors. 4. General Symptoms and Signs of Increased Intracranial Pressure. 5. Cerebral Tumors, Focal Signs and Symptoms. 6. Tumors of Cerebellum, Pons and Medulla. 7. Diseases of the Pituitary Gland. 8. Differential Diagnosis Between Brain Tumors and Other Conditions. 9. Anesthesia. 10. Operative Technique and General Discussion of Neurosurgical Methods. 11. Cranial Cases with the Operative Procedures that were Employed. 12. Spinal and Peripheral Nerve Operations. 13. Closure of Wounds. 14. Post-operative Care. 15. Review of Neurological Surgery and a Glimpse into its Future.

Developments in the field of neurosurgery during the last 25 years have been largely the contribution of the American school led by Cushing, whose co-workers and pupils have, in turn, established centres for neurological surgery in many different parts of the world. Whilst the names Cushing, Dandy, Penfield and Bailey are the best known, Ernest Sachs is among the doyens of the specialty and, as far back as 18 years ago he published the first edition of this book on the *Diagnosis and Treatment of Brain Tumours*, which now appears in its second edition.

It may be questioned whether the opening chapter on surgical anatomy and physiology is necessary in a book such as this. It is somewhat sketchy and incomplete and the student of neurosurgery will require to do much supplementary reading to acquire the necessary basic knowledge.

In dealing with the methods of examination, recently introduced investigatory procedures are mentioned, but certainly do not receive the attention they merit. It would surely not have been difficult, nor would it have required much space, to deal more adequately with electroencephalography in the diagnosis of brain tumours and there is no excuse whatever for the misleading statement that the normal brain produces an alpha rhythm of six per second.

Angiography is so important a subject that it should receive much more than the brief mention given it here. The author quite correctly points out the dangers of lumbar puncture in patients with evidences of increased intracranial pressure and he would have done well to stress the even greater dangers inherent in performing encephalography on patients suspected of cerebral tumour in whom the only evidence of increased intracranial pressure is a raised cerebrospinal fluid pressure.

The chapter on surgical pathology follows the now usually accepted pattern and the only comment one can make is in regard to the incidence of certain types of tumour as stated by the author; surely 45% is much too high an incidence for meningiomas, even though it may include all types of fibroblastic tumours.

The chapter on the symptoms of increased intracranial pressure is a good one and he mentions the important point that whilst bilateral sixth nerve palsy is evidence of a focal pontine lesion, in which case it is fixed and invariable, it can also occur as evidence of general increased pressure, when it is very variable and tends to fluctuate. The subsequent chapters on the focal signs and symptoms are, perhaps, the best in the book.

There are many case protocols, good pictures and the important features are fully explained. Importance is rightly laid on the fact that in many cases evidence of increased intracranial pressure may not occur or may be very late in the history of the case and the experienced clinician will not be misled by this but will proceed with the necessary investigations where these are indicated.

The neurosurgeon will find in the chapters on operative technique and the post-operative care of the patient much of value; it is here, especially, that the author's vast experience of his subject is well evidenced.

As is the case in other special fields of surgery, neurosurgery is on the march and one can confidently look forward to further progress in this most skilful art. This book is a useful publication which the neurosurgeon, the neurologist and the post-graduate student will find a necessary addition to his library. The publishers have done their work well and only four printers' errors were observed in a book of 552 pages.

ANATOMY

A Method of Anatomy. By J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin.). (Pp. 852 + xxiv. With illustrations. 38s. 6d.) London: Baillière, Tindall & Cox. Fourth Edition. 1948.

Contents: 1. General. 2. The Upper Limb. 3. The Abdomen. 4. The Perineum and Pelvis. 5. The Lower Limb. 6. The Thorax. 7. The Head and Neck. 8. Miscellaneous. Index.

This is a regional account of anatomy which employs almost throughout the Birmingham revision of the B.N.A. terminology.

Professor Grant has certainly succeeded in his object, which was to make anatomy 'rational, interesting and of direct application to problems of medicine and surgery'.

The essential principles followed are to give the student the opportunity to correlate facts and so minimize the amount of sheer memory work which the study of anatomy must necessarily imply.

The approach is regional in the sense that the account of a particular portion is built up round the dominant characteristics for the region. Bones, for example, are not described as a thing apart. They are related to the surrounding soft parts and the joints into which they enter.

The emphasis on function makes this a valuable account which more than fulfils the requirements of the modern medical student. The book should prove a useful practical reference text as well.

CORRESPONDENCE

MEDICAL TREATMENT OF COLLEAGUES

To the Editor: With reference to the recent correspondence under this heading, I prefer that a charge be made by the practitioner providing the service. The charge should include out of pocket expenses and a fee for the services rendered. The patient feels more at his ease when he can give recompense for the care and consideration shown by the colleague.

My view is based on the experience of having never been presented with an account for medical service rendered either to myself or my family.

'Black Eye'.

17 January 1950.

THE LATE DR. MACVICAR

To the Editor: For a number of years Dr. Macvicar and I were close friends, and we discussed weekly medical, educational and social problems of common interest. Among the many outstanding physicians whom I had the privilege to meet in my life, none could compare with Dr. Macvicar. Three features combined to make him a unique figure: his extraordinary intelligence, his moral stature and his conviction that the function of science is to serve society.

I first consulted him in connection with my Transkei survey 10 years ago. He helped me to check facts, drew my attention to significant conclusions which the material warranted and finally impressed upon me that any regional planning scheme must be based upon a balanced pattern of human rights as well as of human obligations.

Later I suggested to him that he undertake, for our journal *Manpower*, a comparative survey of vocational and technical education for Africans in the various territories of this continent. This he did, making use of the facilities of the Christian Council with which he was closely associated. He spent much time on this survey, which was printed first in our journal and subsequently distributed as a special booklet by the Race Relations Institute. To our surprise he found that though very much remains to be done, South Africa leads the continent in vocational and technical education for Africans.

The last major research project in which we collaborated was a repetition of the comprehensive survey of tuberculosis among South African natives which he had originally undertaken in the early years of this century. This survey, which has not yet been published, revealed that in the large geographical region surveyed by Dr. Macvicar there has, in the past half century or so, been no increase in the incidence of tuberculosis.

Among the numerous, valuable scientific contributions which Dr. Macvicar has made, none has impressed me more than his study of superstition among natives, published some years ago at Lovedale. I wish every teacher and every administrator in this country would read it. It displays an astounding insight into the mind of the African, and it offers interesting and important suggestions.

Dr. Macvicar was one of my most beloved friends. He was learned, gentle and helpful. Often he was critical, though his criticism was invariably constructive. He was a good teacher and, like many others, I am immeasurably indebted to him. South Africa is the poorer for the passing of this good, unselfish and honest man.

Ernst Jokl.

Pretoria.
20 January 1950.

COLE'S OPERATIVE SURGERY

To the Editor: I read with great interest the review in the *Journal* of Cole's *Operative Surgery*.

There occurs one rather unkind statement unfounded on fact that I cannot allow to go unchallenged. Because the author gives more particulars about the operation for common bile duct reconstruction than does the average surgical textbook, your reviewer arrives at the conclusion that in the United States a lot of bad gall bladder surgery is being done. Because an author goes into detail into a practically never mentioned constructive operation, a sinister motive is sought for as the underlying cause. In doing so, an undeserved slur is cast on American surgery. May I remind your reviewer of the old saying that 'He who has lost no case yet has not yet performed a hundred operations?' Does anybody think less of a surgical giant like Thorek who has written a bulky volume on surgical mistakes? On the contrary, only a great man could do a thing like that.

May I finally remind your reviewer that he need not fear for American surgery—abdominal, plastic, urogenital or any other branch. He need only bear in mind that it was a Yankee, Dr. Long, who initiated ether as an anaesthetic agent; that the first successful oophorectomy was done by a Yankee backvelder, Ephraim McDowell, that Fitzgerald of Boston in 1896 popularized appendectomy (England waited for King Edward's appendiceal abscess in 1907); that the Mayo brothers were the first regularly to practise cholecystectomy; that New Yorker Howard Lilienthal was the pioneer of modern chest surgery; that Harvey Cushing of Boston laid the foundations for modern brain surgery; that Chevalier Jackson is the father of bronchoscopy; that 'blue babies' and essential hypertensive cases are to-day saved through the operative techniques worked out by Blalock and Smithwick; and finally that to-day none of these formidable operations could have been performed with impunity were it not for the pioneer research done on shock by that other American surgical giant, my late teacher and friend, George Crile, who already has left us, but whose work is being carried on by his son, George Crile, Jr., a no less greater teacher and surgeon than his father.

Willem P. Steenkamp, Jr.

S.A. Mutual Building,
Darling Street,
Cape Town.
20 January 1950.

COMPULSORY INTERNSHIP

To the Editor: As some of the recent graduates who have been successful in obtaining employment as interns, either suitable or otherwise, we would like to state our views on the matter.

While in agreement basically with the scheme for compulsory internship and realizing that the Medical Council is doing its best to create positions for those as yet unplaced, we feel that the positions being created, e.g. in Mental and Tuberculosis Institutions, are completely unsuitable for an initial internship. Furthermore, many who have accepted positions in smaller hospitals have only done so because they are unable to register with the Medical Council until they have definite employment and have thus accepted these positions although realizing they are unsuitable.

The scheme has failed firstly, because it has not succeeded in placing everybody as yet. Secondly, and perhaps more important, many have accepted posts which are undesirable and which they feel will not benefit them; thirdly, it has created many problems for married ex-servicemen and women graduands who are unable to accept employment away from their home towns.

Thus, owing to the fact that the scheme has failed so badly this year, we feel we are being deprived of our right of registration on both moral and probably also on legal grounds (see the interview with a barrister in the *Cape Times* of 21 January 1950). In the light of these circumstances it would be reasonable for the Medical Council to reconsider its decision with regard to registration.

'Fortunate Interns'.

21 January 1950.